IT IS CLAIMED:

1. A peptide selected from the group consisting of δV1-1 (SEQ ID NO:4), δV1-2 (SEQ ID NO:5), ψδRACK (SEQ ID NO:6), δV1-5 (SEQ ID NO:7), and derivatives and fragments thereof.

2. The peptide of claim 1 wherein the derivative is selected from the group consisting of δV1-1 derivatives identified as SEQ ID NOS:34-48.

3. The peptide of claim 1 wherein the derivative is selected from the group consisting of δV1-2 derivatives identified as SEQ ID NOS:65-71.

4. The peptide of claim 1, wherein the derivative is selected from the group consisting of ψδRACK derivatives identified as SEQ ID NOS:11-19, 22-33.

5. The peptide of claim 1, wherein the fragment has the sequence selected from the group identified as SEQ ID NOS:49-64.

6. The peptide of claim 1, wherein the fragment has the sequence selected from the group identified as SEQ ID NO:20 and SEQ ID NO:21.

7. The peptide according to claim 1, wherein said peptide is recombinantly produced.

8. The peptide of claim 1, wherein the peptide is chemically synthesized.

9. The peptide of claim 1, wherein the peptide is encoded by a polynucleotide.

10. The peptide of claim 1, wherein the peptide is linked to a moiety effective to facilitate transport across a cell membrane.

11. The peptide of claim 10, wherein the peptide is linked to a moiety selected from the group consisting of a Tat-derived peptide, an Antennapedia carrier peptide, and a polyarginine peptide.

12. The peptide of claim 1 joined to a second peptide to form a fusion peptide.

13. A method of reducing ischemic injury to a cell or a tissue exposed to hypoxic conditions, comprising

administering to the cell or tissue an amount of a δPKC antagonist.

14. The method of claim 13, wherein said administering occurs prior to exposing the cell or tissue to said hypoxic conditions.

15. The method of claim 13, wherein said administering occurs during exposing the tissue to said hypoxic conditions.

16. The method of claim 13, wherein said administering occurs after exposing the tissue to said hypoxic conditions.

17. The method of claim 13, wherein said administering includes administering an antagonist selected from the group consisting of δV1-1 (SEQ ID NO:4), δV1-2 (SEQ ID NO:5), δV1-5 (SEQ ID NO:7), and derivatives and fragments thereof.

18. The method of claim 17, wherein said administering includes administering a δV1-1 derivative antagonist selected from the group consisting of SEQ ID NOS:34-48.

19. The method of claim 17, wherein said administering includes administering a δV1-2 derivative antagonist selected from the group consisting of SEQ ID NOS:65-71.

20. The method of claim 17, wherein said administering includes administering a peptide fragment selected from the group of peptides identified as SEQ ID NOS:49-64.

21. The method of claim 13, wherein said administering includes administering a peptide linked to a moiety effective to facilitate transport across a cell membrane.

22. The method of claim 13, wherein said administering includes administering a peptide linked to a moiety selected from the group consisting of a Tat-derived peptide, an Antennapedia carrier peptide, and a polyarginine peptide.

23. The method of claim 13, wherein said administering is by infusion through coronary arteries to an intact heart.

24. A method of reducing damage to a cell or tissue from a hypoxic event due to stroke, comprising

administering to the cell or tissue an amount of a δPKC antagonist.

25. The method of claim 24, wherein said administering occurs prior to said hypoxic event.

26. The method of claim 24, wherein said administering occurs during exposing the tissue to said hypoxic event.

27. The method of claim 24, wherein said administering occurs after exposing the tissue to said hypoxic event.

28. The method of claim 24, wherein said administering includes administering an antagonist selected from the group consisting of δV1-1 (SEQ ID NO:4), δV1-2 (SEQ ID NO:5), δV1-5 (SEQ ID NO:7), and derivatives and fragments thereof.

29. The method of claim 28, wherein said administering includes administering a δV1-1 derivative antagonist selected from the group consisting of SEQ ID NOS:34-48.

30. The method of claim 28, wherein said administering includes administering a δV1-2 derivative antagonist selected from the group consisting of SEQ ID NOS:65-71.

31. The method of claim 28, wherein said administering includes administering a peptide fragment selected from the group of peptides identified as SEQ ID NOS:49-64.

32. The method of claim 24, wherein said administering includes administering a peptide linked to a moiety effective to facilitate transport across a cell membrane.

33. The method of claim 24, wherein said administering includes administering a peptide linked to a moiety selected from the group consisting of a Tat-derived peptide, an

Antennapedia carrier peptide, and a polyarginine peptide.

34. A method of enhancing damage to a cell exposed to hypoxic conditions, comprising administering to the cell an amount of an isozyme-specific δPKC agonist.

35. The method of claim 34, wherein the agonist is ψδRACK identified as SEQ ID NO:6, a derivative or a fragment thereof.

36. The method of claim 34, wherein the agonist is a peptide selected from the group consisting of SEQ ID NOS:11-19, and SEQ ID NOS:22-29.

37. The method of claim 34, wherein the agonist is a peptide selected from the group consisting of SEQ ID NOS:20-21.

38. The method of claim 34, wherein the cell is a tumor cell.

39. The method of claim 34, wherein said agonist is linked to a moiety effective to facilitate transport across a cell membrane.

40. A method of identifying a compound effective to induce protection of a cell from hypoxic or ischemic damage, comprising

contacting a δPKC peptide containing a δRACK binding site with a δPKC antagonist peptide with the δRACK binding site in the presence and absence of said test compound, and

identifying said test compound as being effective to induce protection if (i) binding in the presence of the test compound is decreased relative to binding in the absence of the test compound, or (ii) catalytic activity of the test compound is increased relative to activity in the absence of the test compound.

41. The method of claim 40, wherein said contacting includes contacting a δPKC peptide selected from the group consisting of δV1-1 (SEQ ID NO:4), δV1-2 (SEQ ID NO:5), δV1-5 (SEQ ID NO:7, and fragments and derivatives thereof.

42. The method of claim 39, wherein said contacting includes contacting with δPKC antagonist peptide selected from the group consisting of SEQ ID NO:34-48.

43. The method of claim 39, wherein said contacting includes contacting with δPKC antagonist peptide selected from the group consisting of SEQ ID NO:49-64.

44. The method of claim 39, wherein said contacting includes contacting with δPKC antagonist peptide selected from the group consisting of SEQ ID NO:65-71.

45. The method of claim 39, wherein said δPKC peptide containing a δRACK binding site is identified as SEQ ID NO:72.

46. A method of identifying a compound effective to enhance hypoxic or ischemic damage in a cell, comprising

contacting a ψδRACK agonsit peptide with a δPKC peptide containing a RACK binding site in the presence and absence of a test compound, and

identifying said test compound as being effective to enhance ischemic damage if (i) binding in the presence of the test compound is decreased relative to binding in the absence of the test compound, or (ii) the catalytic activity of the δPKC in the presence of the test compound is increased relative to the catalytic activity in the absence of the test compound.

47. The method of claim 46, wherein said contacting includes contacting with a ψδRACK peptide selected from the group consisting of SEQ ID NO:6, fragments, and derivatives thereof.

48. The method of claim 46, wherein said contacting includes contacting with a ψδRACK peptide having a sequence selected from the group of peptides identified as SEQ ID NOS:11-19 and SEQ ID NOS:22-29.

49. The method of claim 46, wherein said contacting includes contacting with a ψδRACK peptide fragment having a sequence selected from the group of peptides identified as SEQ ID NOS:20-21.

50. A method of providing protection to tissue from damage caused by an ischemic or hypoxic event, comprising

administering to the tissue a peptide selected from the group consisting of SEQ ID NO:4, SEQ ID NO:5, SEQ ID NO:7, derivatives and fragments thereof.

51. The method of claim 50, wherein said administering includes administering a peptide derivative selected from the group consisting of δV1-1 derivatives identified as SEQ ID NO:34-47.

52. The method of claim 50, wherein said administering includes administering a peptide derivative selected from the group consisting of δV1-2 derivatives identified as SEQ ID NO:65-71.

53. The method of claim 50, wherein said administering includes administering a peptide fragment selected from the group consisting of peptides identified as SEQ ID NOS:49-64.

54. The method of claim 50, wherein said administering includes administering a peptide linked to a moiety effective to facilitate transport across a cell membrane.

55. The method of claim 54, wherein said administering includes administering a peptide linked to a moiety selected from the group consisting of a Tat-derived peptide, an Antennapedia carrier peptide, and a polyarginine peptide.

56. The method of claim 50, wherein said administering includes administering the peptide by a route selected from the group consisting or intraveneous, parenteral, subcutaneous, inhalation, intranasal, sublingual, mucosal, and transdermal.

57. The method of claim 50, wherein said administering occurs during reperfusion.

58. The method of claim 50, wherein said administering is effective to provide protection against ischemia of a tissue selected from the group consisting of brain, heart, eye, and kidney.